No.	Age	Sex	Diagnosis	Dosage	Length of treatment	Day of white cell count in brackets				
1	64	M	C.B.	3 b.d.	14 days	(1) WBC 5100 N 3400	(14) 3500 2000			
2	17	M	В	3 b.d.	14 days	WBC 4800 N 2800	(14) 3800 2400	(28) 4500 2000	•	
3	44	М	C.B A	3 b.d.	7 days	WBC 5000 N "normal"	(7) 3000 1100			
4	73	F	C.B.	2 b.d.	7 days	WBC 5600 N 3700	(7) 3000 1600	(14) 6200 3200		
5	68	М	C.B.	3 b.d.	7 days	WBC 3800 N 2500	(7) 3600 1900	(21) 4600 2200		
6	64	F	C.B.	2 b.d.	7 days	WBC 4700 N 3000	(7) 4000 2400			
7	62	М	C.B.	2 b.d.	28 days 10 days off 5 days	WBC 9800 N 7000	(28) 7700 2700	(43) 5600 2600	(50) 4400 1700	(57) 11,000 8800
8	51	М	C.B.	3 b.d.	7 days*	(1) WBC 6400 N "normal"	(7) 4600 2200			

*14 days treatment with 2 tabs. twice daily completed 2 weeks previously.

A = asthma. B = bronchitis. C.B. = chronic bronchitis. WBC = total white count. N = neutrophils.

1.6 g., or trimethoprim 480 mg. with sulphamethoxazole 2.4 g., usually for 7 or 14 days. White cells were counted on the first day of treatment and again on the last day. lower limit of normal for the white cell count was taken as 4,000 per cu. mm. and for neutrophils 2,500 per cu. mm.5

Out of the 32 patients there were eight whose neutrophil counts were below normal at the end of treatment; seven of these also had an abnormally low total white cell count. These abnormalities seemed to be significant in four patients (1-4; Table), but the other four abnormal counts could have been due to experimental error. Twenty-four of the remaining 28 total white counts fell after treatment, though not dropping below normal levels; initially eight of these had shown a leucocytosis and 16 had been normal. affected group showed no important difference in age, sex, dosage, or length of treat-ment. Patients 7 and 8 had had a previous course and patient 4 was given a subsequent course without adverse effect on the white cells on these occasions.

The return of the white cell count to normal was prompt in two patients but incomplete in one patient fourteen days after treatment finished, though ultimately complete in all. It must be stressed that there was no clinical evidence of illness attributable to the neutropenia, and that there was no evidence of its having affected the patients

I wish to thank Dr. M. Caplin, of the London Chest Hospital, who initiated the study and Dr. I. Lenox-Smith, of Roche Products Limited, who supplied the Bactrim.

-I am, etc.,

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was a typical generalized erythrodermia with extensive superficial desquamation, and in addition there were many excoriated nodular lesions. The only other abnormal physical finding was a mild generalized lymphadenopathy. Sigmoidoscopy revealed mild mucosal oedema in the distal 10 cm. of the bowel with no ulceration or contact bleeding; mucosal scrapings were taken and immediately examined microscopically. They showed haematophagous Entamoeba histolytica trophozoites. His amoebiasis was treated with metronidazole. No underlying cause was found for his erythrodermia, which was treated symptomatically with some difficulty.

There can be little doubt that this patient's recent diarrhoeal illness was acute amoebic dysentery. Although he had no bowel symptoms when first seen by us, the finding of haematophagous trophozoites indicates that the amoebiasis was still in an invasive phase. We presume that prior to the steroid therapy this patient was an asymptomatic amoebic cyst-passer. It should be pointed out that a cyst-passer who develops diarrhoea for any reason, including that following the administration of a purgative may sometimes show small trophozoites in the stool. These are never haematophagous and are referred to as the minuta forms of E. histolytica. However, this does not apply to our patient.

We are grateful to Professor A. W. Woodruff and Dr. H. A. K. Rowland for permission to publish this case report.

-We are, etc.,

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Hospital Planning

SIR,-Professor E. D. Acheson's informative article (21 June, p. 750) on Southampton Medical School is illustrated by a photograph of the plan of a new Southampton General Hospital complex. It contains ward blocks for 1,300 patients, together with the ancillary services and teaching facilities. It contains four nine-storey residential blocks and three three-storey residential blocks. It contains the administrative offices, the laundry, the nurses' home, etc. It contains the outpatient services. It will certainly contain a very large number of people, many of whom, patients and others, will go to and from the hospital each day.

It contains no provision whatever for car parking.

The planning of this complex has occupied the time of a great number of people for some time past, and will continue to do so for some time to come. Integrated planning has been done in great detail, down to the size and shape of the smallest rooms. Most of those concerned have at one time or another spoken of the need for very large car-parking facilities, but this is always immediately met by the statement that the Ministry will make no provision for this.

The fundamental basic object in building this complex is to provide a meeting-place for patients, doctors, nurses, teachers, students, administrators, and lay workers. They will be coming and going at all times of the day and night, seven days a week. No matter what public transport services may be available, very many will require

Amoebic Dysentery Precipitated by Corticosteroids

SIR,-In our paper about relapsing amoebic colitis exacerbated by corticosteroids (7 June, p. 613) we pointed out that any patient who develops diarrhoea, particularly those who have resided in the tropics, while on corticosteroids prescribed for any condition should be carefully investigated for amoebiasis. Since the time of writing we have encountered a patient who well illustrates this problem.

An Englishman, aged 57, who had lived in Ceylon for 30 years, returned to Britain about six years ago, since when he has visited Ceylon once for a five-week period in 1967. He was treated for "dysentery" during his first voyage home. In November 1967 he developed a mild pruritic dermatosis on his legs and forearms,

which gradually became worse and generalized. In February 1969 he consulted a dermatologist who diagnosed dermatitis herpetiformis and he started treatment with dapsone and oral antihistamines. After two weeks, as there was no improvement, he was given prednisolone 15 mg. twice daily for three weeks. Within 48 hours of starting steroid therapy he developed diarrhoea with fresh blood and mucus; he was passing seven stools each day. These symptoms per-sisted until one week after the steroids were stopped. Dapsone therapy was then restarted, on this occasion at a higher dosage, but he developed oedema of the face, cervical lymphadenopathy, and an aggravation of his skin

When first seen by us on 14 April he complained of intense pruritus, but the diarrhoea had completely subsided. On examination there